Amendments to the Specification:

Please delete the existing title and replace it with the following rewritten title:

- - **A METHOD FOR MODULATING THE ACTIVITY OF A PEPTIDE TRANSFERASE CENTER** - -

Please insert the following paragraph immediately after the title of the specification:

- - **CROSS REFERENCE TO RELATED APPLICATIONS** - -

The present application is a divisional of U.S. Application No. 08/724,992, filed October 4, 1996, which claims the benefit of U.S. Provisional Application No. 60/005,041, filed October 6, 1995, the contents all of which are incorporated herein by reference. - -

Please delete the existing sequence listing and replace it with the substitute sequence listing enclosed herewith.

Please replace the paragraph beginning at page 7, line 19 with the following rewritten paragraph:

- - **FIGURE 1**. The "integrated model" of -1 ribosomal frameshifting. (A) depicts how -1 ribosomal frameshifting is related to the stages of translation elongation. Translation of the theoretical base sequence of SEQ ID NO:6 is depicted. (B) depicts how Tyl mediated +1 ribosomal frameshifting is related to translational elongation. Translation of the theoretical base sequence of SEQ ID NO:15 is depicted. - -

Please replace the paragraph beginning at page 8, line 18, with the following rewritten paragraph:

- - FIGURE 4. Identification and characterization of the lesion and the *mof4-1* allele. Hybrid genes between the wildtype *UPF1* and the *mof4-1* alleles schematically represented in panel A were constructed, transformed into a *upf1* Δ strain and CYH2 precursor abundance was determined by RNA blotting analysis as described in Figure 1. An autoradiogram of this analysis is shown in panel B. The black rectangle in panel A represents sequences from the wildtype *UPF1* gene while the hatched rectangle represents sequences from the *mof4-1* allele. The cysteine-rich region of the *UPF1* gene is represented by gray rectangle in the wild type *UPF1* gene. A sequence from the wildtype *UPF* gene (SEQ ID NO:7) is depicted. A mutation in the gene accounts for the *mof4-1* allele. The dark vertical line represents the location of the mutation within the *mof4-1* allele. The *mof4-1* allele was sequenced and the sequence change is shown. For each hybrid allele shown in Panel A two identical constructs were prepared from different PCR reactions and are designated with the subscript 1 or 2 in panel B. The restriction endonucleases represented in panel A: E1 ~~(EcorR1)~~(EcoRI), Bst(BstX1), Asp(Asp718), B1(BamH1).

Please replace the paragraph at page 9, line 1, with the following rewritten paragraph:

- - FIGURE 5. A) cloning strategy for cloning *mof2-1*. B) Sequence analysis to identify the mutation in the *mof2-1* allele. Figure 5B depicts a comparative alignment of *SUI1* homologues from Human (SEQ ID NO:1), Mosquito (SEQ ID NO: 2); Rice (SEQ ID NO: 3); Yeast (SEQ ID NO:4); and Methanococcus sp (SEQ ID NO:5). - -

Please replace the paragraph at page 28, line 1, with the following rewritten paragraph:

- - Additionally, the frameshift or mRNA decay protein-encoding nucleic acid sequence can be mutated *in vitro* or *in vivo*, to create and/or destroy translation, initiation, and/or termination sequences, or to create variations in coding regions and/or form new restriction endonuclease sites or destroy preexisting ones, to facilitate further *in vitro* modification. Preferably, such mutations enhance the functional activity of the mutated frameshift or mRNA decay protein gene product. Any technique for mutagenesis known in the art can be used, including but not limited to, *in vitro* site-directed mutagenesis (Hutchinson, C., et al., 1978, J. Biol. Chem. 253:6551; Zoller and Smith, 1984, DNA 3:479-488; Oliphant et al., 1986, Gene 44:177; Hutchinson et al., 1986, Proc. Natl. Acad. Sci. U.S.A. 83:710), ~~use of TAB® linkers (Pharmacia), etc.~~ insertion of small oligonucleotide linkers (TAB® linkers from Pharmacia), etc. PCR techniques are preferred for site directed mutagenesis (see Higuchi, 1989, "Using PCR to Engineer DNA", in *PCR Technology: Principals and Applications for DNA Amplification*, H. Erlich, ed., Stockton Press, Chapter 6, pp. 61-70).